A Study of Histopathological changes in stomach wall at sites other than the ulcer site in peptic ulcer disease and its association with H.pylori

Dissertation submitted to

THE TAMILNADU

DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI – 600 032

with fulfillment of the Regulations

for the Award of the Degree of

M.S. GENERAL SURGERY

(BRANCH – I)

DEPARTMENT OF SURGERY

STANLEY MEDICAL COLLEGE

CHENNAI – 600 001

APRIL – 2013

CERTIFICATE

This is to certify that this dissertation in **"A Study of Histopathological changes in stomach wall at sites other than the ulcer site in peptic ulcer disease and its association with H.pylori"** is a work done by **Dr. K.DALTON JEBARAJ** under my guidance during the period 2010-2013. This has beensubmitted in partial fulfilment of the award of M.S. Degree in GeneralSurgery (Branch – I) by The Tamilnadu Dr. M.G.R. Medical University,Chennai – 32.

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DECLARATION

I **Dr. K.DALTON JEBARAJ** Solemnly declare that the dissertation titled **"A Study of Histopathological changes in stomach wall at sites other than the ulcer site in peptic ulcer disease and its association with H.pylori"** is abonafide work done by me during the period of Between January 2012 to November 2012 at Government Stanley Medical College and Hospital, under the expert guidance of **Prof.G.MUTHUKUMARAN, M.S.,** and **Prof. Dr.T.S.JAYASHREE, D.G.O., M.S.,**unit chiefs of Department of Surgery, Government Stanley Medical College and Hospital, Chennai.01.

This dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University, in partial fulfilment of the rules andregulations for the M.S. degree examinations in GeneralSurgery to be held in April 2013.

Dr. K.DALTON JEBARAJ

Place: Chennai-1

Date :

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INTRODUCTION

Peptic ulcer disease is a common ailment in patients suffering from symptoms of dyspepsia. Duodenal ulcers nearly constitute one third of all cases of peptic ulcer disease .It is characterised by a defined defect in the mucosa which extends into muscular propria as well.

The common causes of PUD are NSAID abuse , decreased mucosal resistance and H.pylori infection.H.pylori infection and hyperchlorhydria can induce stomach and duodenum. It has to be documented whether H.pylori produces any change in stomach wall other than the ulcer site. Infection by H.pylori can occur in gastric mucosal surface as well as mucosa in proximal duodenum.

Recent studies ,plan to eradicate H.pylori, in an attempt to heal peptic ulcer disease have given promising results and proves a clear correlation between Helicobacter pylori infection and peptic ulcer diseases.

AIMS AND OBJECTIVES

1. To study the changes in stomach wall at sites other than the ulcer site in PUD.

2. To correlate the association of stomach wall changes with H.pyloric infection.

REVIEW OF LITERATURE

MURALTO was the one who found out the presence of duodenal ulcers in 1688 in a autopsy

CRISP,presented the first paper regarding perforation in peptic ulcers in 1843.Treatment for perforation had been a great hallmark in general surgery practising since 100 years.

JOHN LYKOUDIS, a physician from Greece in 1958 treated stomach ulcers with medications

H.PYLORI was discovered by scientists from Australia named ROBIN WARREN and BARRY J MARSHALL in 1982 that it was the reason for ulcers

In 1997 centers for disease control for prevention described the association of H.pylori with gastritis and ulcers

NOBEL PRIZE for H.pylori as a cause for peptic ulcer disease was awarded to professor MARSHALL in 2005. He belonged to KAROLINSKA INSTITUTE IN STOCKHOLM

ANATOMY OF STOMACH

Stomach is the most vascularised part of gastrointestinal tract among all organs. Stomach is divided into

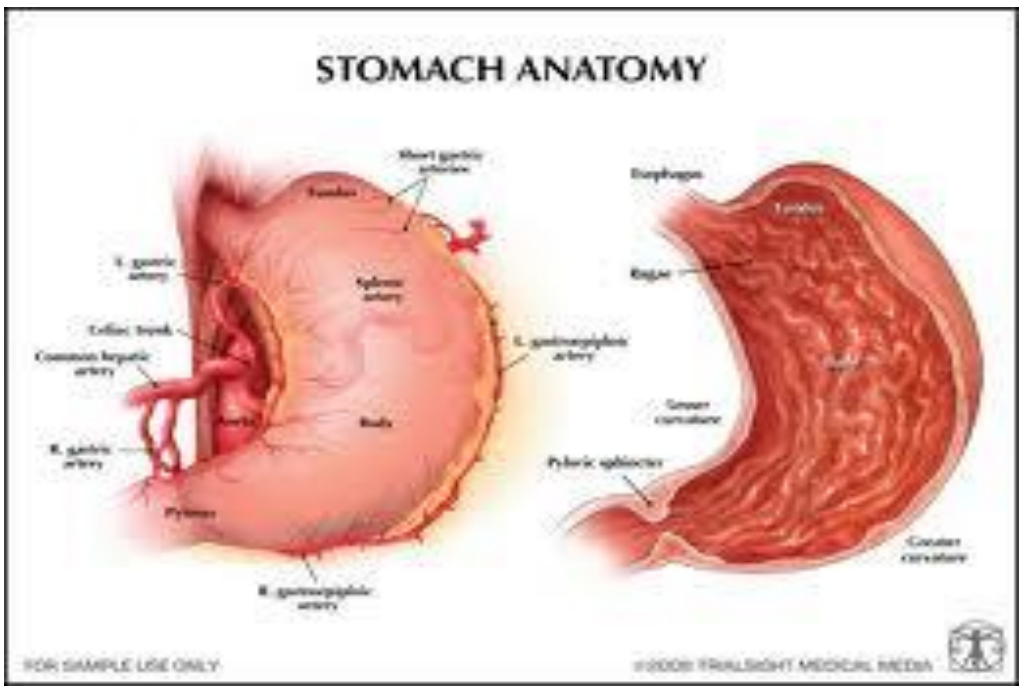
Fundus

Body

Lesser curvature

Greater curvature

Antrum



ARTERIAL SUPPLY

Stomach derives its blood supply mainly from the celiac axis and its branches. The branches are

The right gastric artery

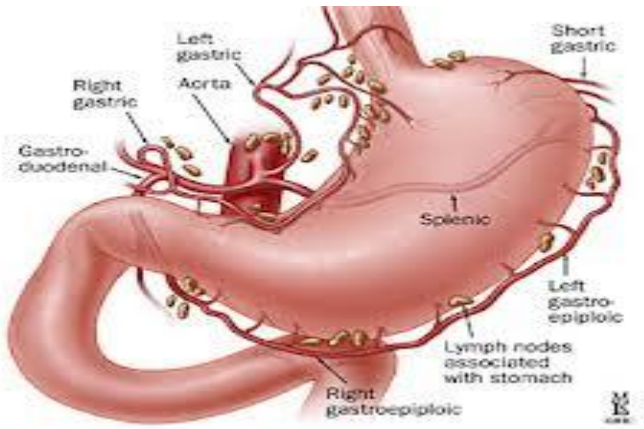
The left gastric artery

The right gastroepiploic artery

The left gastro epiploic artery

The short gastric arteries

The left gastric artery is the largest branch among all arteries. The right and left gartric arteries together form a anastamosis around the lesser curvature . The right and left epiploic arteries form arich arcade around the lesser curvature.The left gatric artery divides into a ascending and descending branch



Venous drainage

1. Left gastric vein

2. right gastric vein

Both veins drain into portal vein.

3.The right gastroepiploic vein drains into the superior mesenteric vein.

4. Left gastroepiploic vein drains into inferior mesenteric vein

Lymphatic drainage

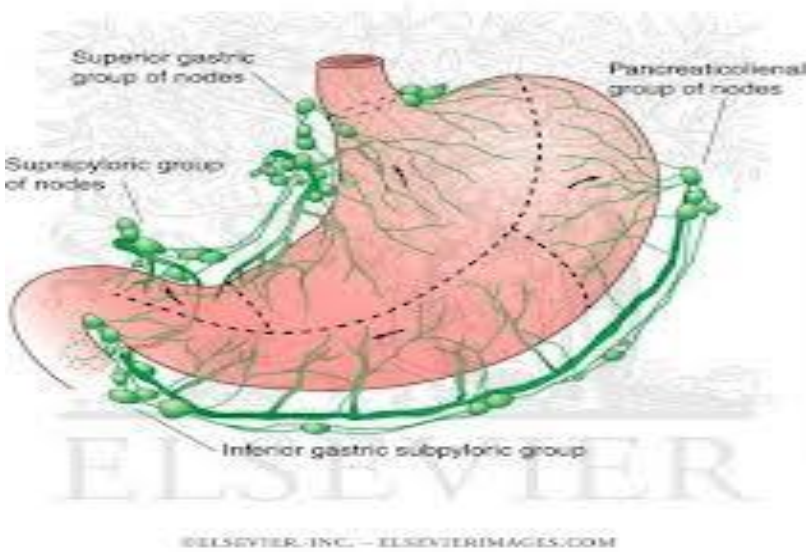
Gastric lymphatics parallel the blood vessels.

1.The cardiac nodes - along left gastric and celiac axis

2.Lesser curvature - nodes along the right gastric

3. Distal greater curvature - nodes along right gastroepiploic vein

4.Proximal greater curvature - nodes along the left gastroepiploic vein in splenic hilum.



LYMHPHATIC DRAINAGE OF STOMACH

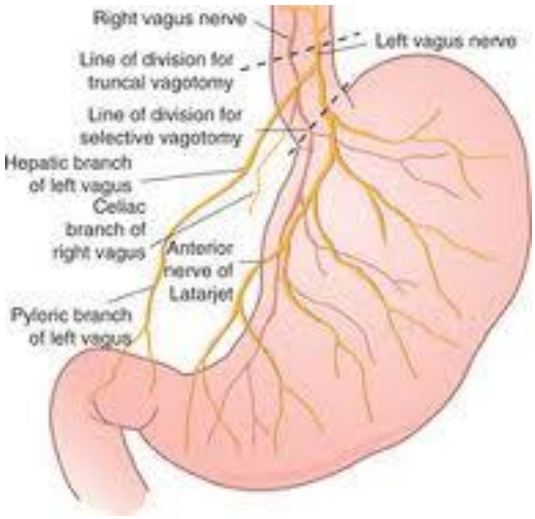
The lymphatic system of stomach is very important as in gastric carcinomas the metastatic spread is mainly through lymphatics . The operative procedures are also mainly concerned about removing the nodes that have been involved in the carcinoma. Nodal spread is very important in determining the prognostic value of the disease.

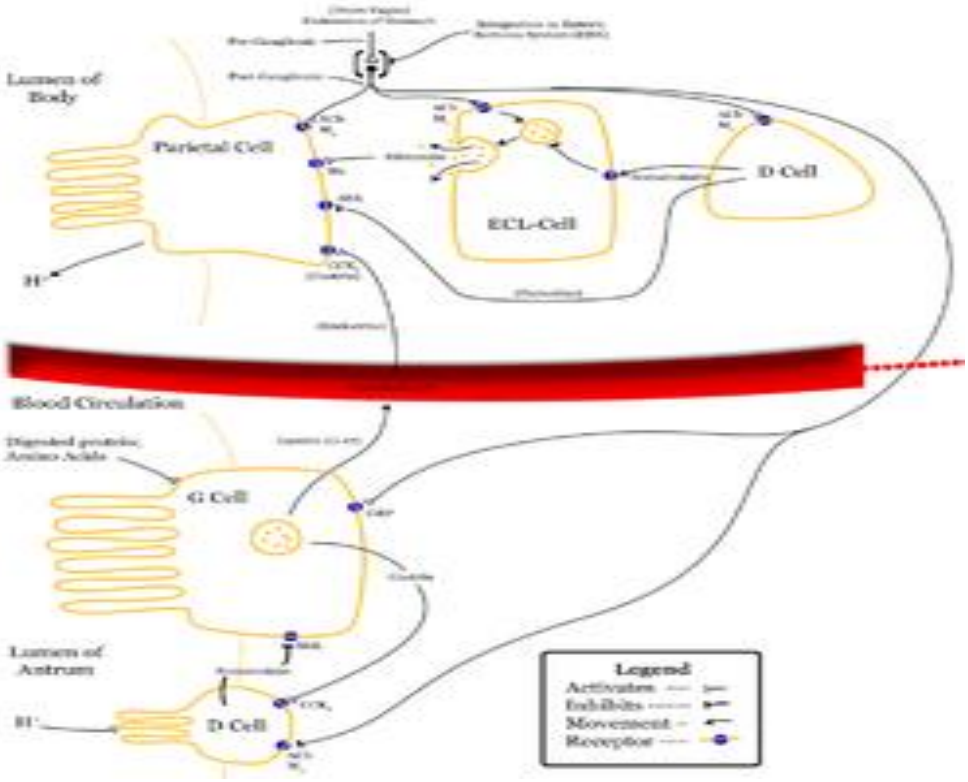
Nerve supply

The extrinsic and intrinsic innervations of the stomach have an important role in gastric secretory and motor function. The vagus provides parasympathetic innervations to the stomach and acetylcholine is the important neurotransmitter.

Near the junction anterior vagus sends branches to liver and continues along the lesser curvature as anterior nerve of LATARJET. These nerves send segmental branches to the body of stomach before they terminate near angularis incisura as crow's pet in antipyloric region. The posterior branch sends branches to posterior fundus and is called CRIMINAL NERVE OF GRASSI AUERBACH'S myenteric plexus and meissner submucosa plexus are found in muscularis and submcosa.

The extrinsic sympathetic nerve supply to the stomach originate at spinal levels T5 to T10 and travels in splanchnic nerve to celiac ganglia





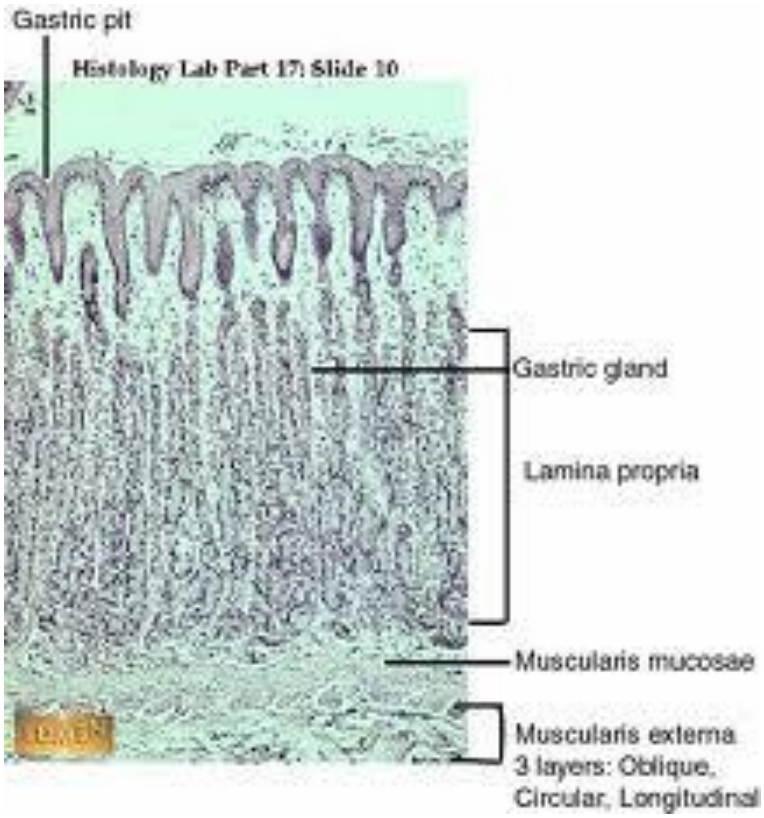
VAGOVAGAL REFLEX

The vagovagal reflex plays a important role in secretion of acid. This reflex is mediated by the local afferent and efferent fibres of this nerve through the dorsal vagal nucleus found in brain. It becomes active when bolus enters the stomach and produces distension of stomach to accomdate the food that enters via esophagus.The stimulation of mechanical receptors found in the body send impulses via afferents and result is activation of post ganglionic muscuranic receptors which secrete acetylcholine that stimulates secretion of hcl by parietal cells and ecl cells to secrete histamine . Histamine is a strong inducer of gastrin

secretion. This vagal stimulation also activates the peptidegenic neurons which are responsible for production of gastrin releasing polypeptide. Delta cells which secrete somatostatin is also inhibited by this vagal stimulation resulting in increased release of gastrin.

The parasympathetic nerves carrying both afferent And efferent nerves recognise the stretch receptors and Osmoreceptors and carry impulses to dorsal vagal complex and return is carried by way of vagal nerves and spread to right 2/3 of transverse colon.

HISTOLOGY



Four distinct layers

1.mucosa

2.submucosa

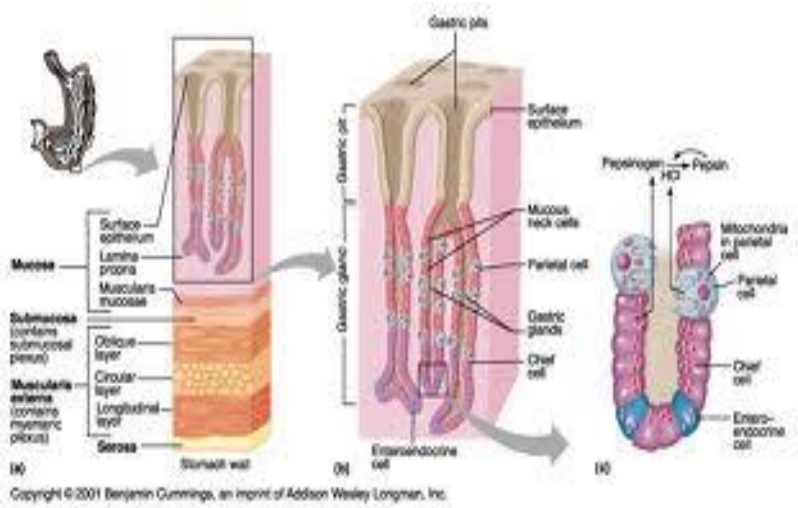
3.muscularis propria

4.serosa

Mucosa is lined by columnar epithelial cells . It is glandular in type. A scanning electron micrograph shows a smooth mucosal plane punctuated by openings of gastric glands. The glands are lined by different types of epithelial cells depending upon their location in stomach. Endocrine cells are present in the gastric glands.

Gastric gland consists of

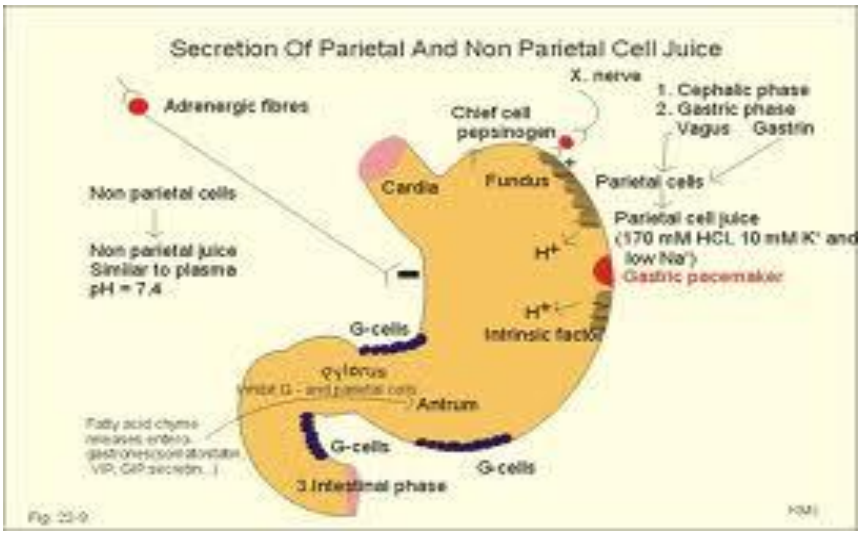
1. Progenitor cells at base of glands.
2. Mucus secreting epithelial cells that secrete bicarbonate and Prevents stomach from injury due to acid, pepsin and ingested irritants.
3. Cheif cells- secrete pepsinogen.
4. Parietal cells- secrete hcl
5. Enterochromaffin cells – secrete histamine.



Physiology of acid secretion

Hcl hastens both physical and biochemical breackdown of ingested food. In acidic environment pepsin and acid facilate proteolysis . gastric acid inhibits ingested pathogen.

The parietal cell is stimulated to secrete acid when its receptor is stimulated by acetylcholine(from vagal fibres, gastrin (from d cells) or histamine (from ecc cells) . the enzyme H+ K+ atpase is the proton pump. It is contained within te intracellular tubulovesicles and is the common pathway for gastric acid secretion. The normal human stomach contain approximately 1 billion parietal cells and entire gastric acid production is proportional to parietal cells in man.



The major role of acid producing capacity of both acetylcholine and gastrin are regulated by histamine release from the mucosal ecl cells. That is why histamine 2 receptor antagonists are such effective inhibitor of acid secretion. The mucosal cells produce somatostatin which is an important regulator of acid secretion.

Somatostatin decreases histamine release from ecl cells and gastric glands. The action of D cells is abolished by pylori infection and this finally lead to increased acid producing response.

The most potent physiologic stimulus for production pepsinogen secretion from chief cells is ingestion of food. Acetylcholine is the important mediator.

Somatostatin decreases pepsinogen secretion. Pepsinogen is broken down to active pepsin enzyme in an acid environment 2.15 ph and inactive at ph>5

Gastric mucosal barrier

Components

1. Mucosa barrier
2. Bicarbonate secretion
3. Epithelial barrier

   Hydrophobic phospholipids

   Tight junction

   Restitution
4. Microcirculation
5. Afferent relay neurons.

Mediators

Prostaglandins

Nitric oxide

Epidermal growth factor

Calcitonin gene related peptide

Hepatocytic growth factor

Histamine

Gastrin releasing peptide

"Barrier destroyers" such as bile or aspirin lead to high diffusion of hydrogen ions from the lumen into the lamina propria and submucosa leading to gross ulceration.

DUODENUM

ANATOMY

Duodenum is the proximal part of small intestine and is continuous proximally with the pylorus. Its forms a c shaped loop around the head of pancreas. In adult length is 30 cm and subdivided into four sections

First

Second

Third

Fourth

The first part of the duodenum is about 5cm long and it is right, upward and backward from the pylorus. The proximal part of duodenum is also referred as duodenal bulb or cap. It is loosely attached to the liver by the hepatoduodenal portion of the lesser omentum. The gastric duodenal artery common bile duct and portal vein is posterior and gall bladder is anterior to it.

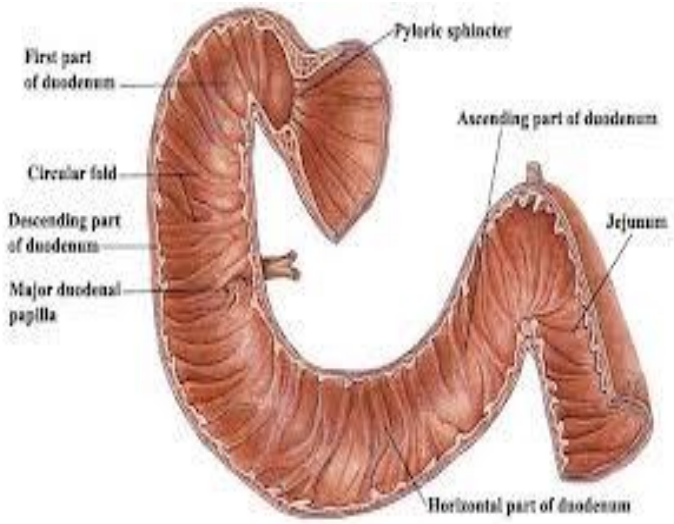


Fig shows the four parts of duodenum and ampulla of vater and the c loop of duodenum and valve conventis

Layers of duodenum

The duodenal wall is made of outer longitudinal and inner circular muscle layer. There are mucosal folds called plicae circularis or vulvulae conniventes.

Duodenal bulk is distinguished radiographically and endoscopically by its smooth mucosa.

Vascular supply

Based on the embryologic origin branches of celiac trunk supply proximal duodenum from the celiac artery arises the common hepatic artery from which gastroduodenal artery rises which gives anterior and posterior branches.

Venous drainage corresponds to arterial supply . The superior pancreaticoduodenal vein passing between duodenum and pancreatic head to enter the portal vein.

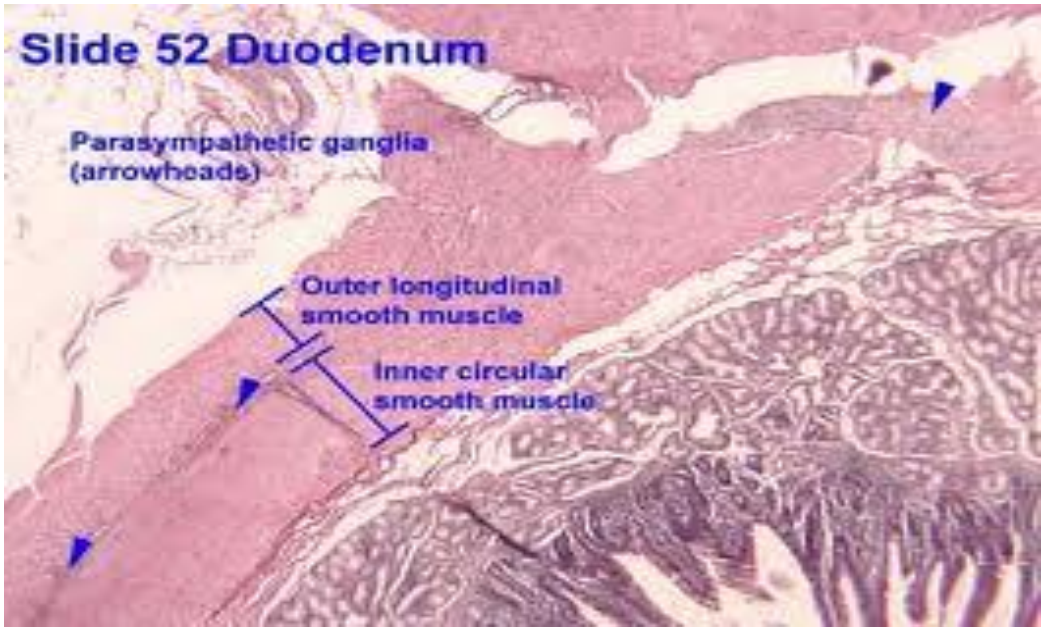
LYMPHATIC DRAINAGE

Duodenal lymphatic drainage also corresponds to the vascular supply. Small anterior and posterior duodenal lymph nodes drain into pancreaticoduodenal nodes. From them lymph drains superiorly into hepatic nodes or interiorly into superior mesenteric nodes located at the origin of superior mesenteric artery.

Duodenal innervations

As stomach duodenal innervations is provided by sympathetic and parasympathetic nervous symptoms. The preganglionic sympathetic nerves cause through the celiac and superior mesenteric ganglion with postganglion nerves entering the duodenal intramural plexus. Afferent plexus accompany the sympathetic neurons primarily carry fibres to visceral pain sensation. Parasympathetic plexus supplied by the hepatic branches of the anterior vagus nerve and mesenteric nerves, synapse with meissner's and auerbach plexus in the duodenum

Histology



The duodenum differ from gastric mucosa with the drainage from gastric glands and pits to mucosa lined with villi surrounded by crypts of lieberkunn and submucosa certain characteristic brunner's gland. A single layer of epithelial cells provide interface between duodenal lumen and mucosa in areas of both villi and crypts deep to epithelial layer are contained absorptive cells, paneth cells secreting apozyme mucosal cells and endocrine cells.

PEPTIC ULCER DISEASE

These are ulcers in the stomach or duodenum which may continue into the deeper layers of stomach wall. They are mainly due to loss of balance between mucosal defences and acid secretion PUD remains a OP diagnosis but number of hospital visits and admissions have decreased markedly in the last three decades due to the invention of H2 receptor d fiboptic endoscopy. However the incidence of emergencies due to peptic ulcer has decreased so dramatically.

The epidemiological changes are usually due to some beneficial effect such as

1. Decreased prevelance of H.pylori infection.

2. Better medical therapy

3.Increased ourtpatiient management

Some appreciating factors are use of NSAIDS and aspirins in aging population with multiple risk factors.

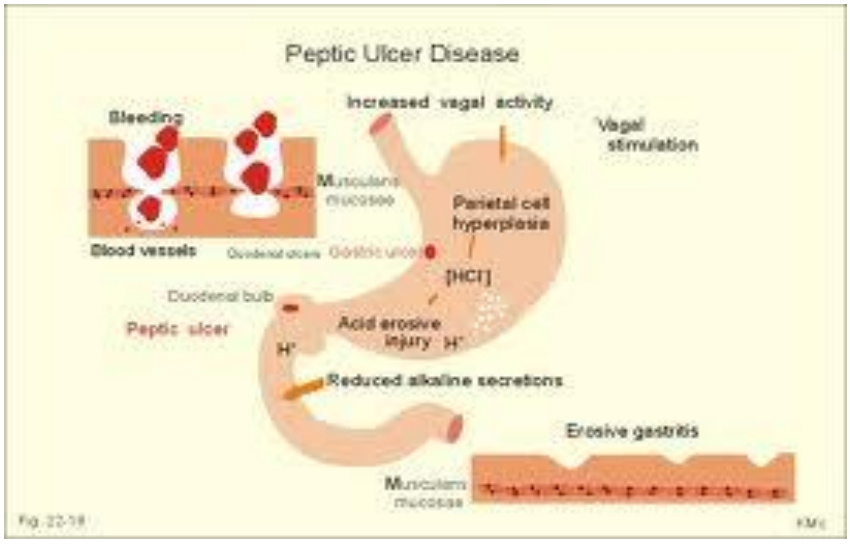
PUD is one of the most common a1 disorders having a prevalence rate of 2% and peaks around usually 70 years of age. The crude mortality rate due to peptic ulcer disease was nearly 1.7 per 1 lakh individuals.

Stomach wall erosions has a increased rate of mortality When compared to duodenal ulcer since it is mainly found in elderly patients. Recent data suggest that there is an increase in admission and mortality in old individuals due to PUD complications of bleeding and perforation. This may be because of dramatic usage of NSAIDS and aspirin in elderly people.

Pathophysiology

A large number of factors contribute to the development of PUD recent studies indicate that most of duodenal and gastric ulcer are produced by H.pylori infection and NSAID ingestion. The final end step is the ulcer formation due to breakage of gastroduodenal mucosal barrier. Suppression of acid is the main form of treatment.

It is proved that h.pylori leads to ulcer formation by increase in acid secretion and damage to mucosa by damaging SEC. Duodenal disease is usually due to high acid peptic action in the mucosa whereas gastric ulcer has been due to weakening of mucosal defences in the face of normal acid activity.



Decreased mucosal defences play an important role in many ulcers

Example: DU ulcer in patients not infected with hpylori individuals on NSAIDS.

Person with category I gastric ulcer with acid decrease in acid secretion High corrosive action of acid may produce ulcer with normal defense mechanism

Example: DU ulcer in zollinger Ellison syndrome

Gastric ulcer in outlet destruction patient, antral stasis and acid hypersecretion

A variety of diseases producing peptic ulcers are

Gastrinomas

Antral cell hyperfunction

Systematic mastocytosis

Trauma

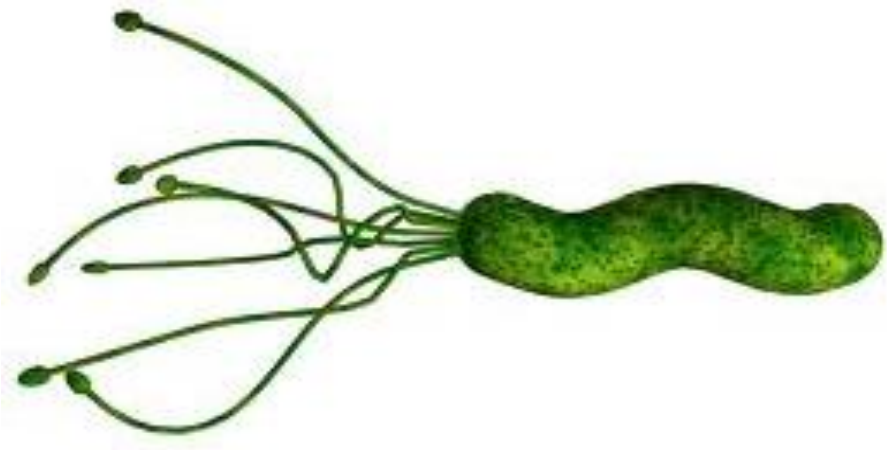
Stress

burns

Helicobacter pylori:

H.pylori has a specialised flagella.It has an enormous amount of urease.This is responsible for survival in the acidic medium of the stomach.Nearly 60% of the population is found to be infected with H.pylori.

The sequence phenomenon of inflammation producing the metaplasia to dysplasia to carcinoma which is observed in oesophagus is found to develop in stomach due to H.pylori infection.The suppression of acid due to ppis and h2a antagonists on these esophagogastric inflammation is largely unknown.H.pylori is found to have a definitive role in the development of gastric lymphoma.



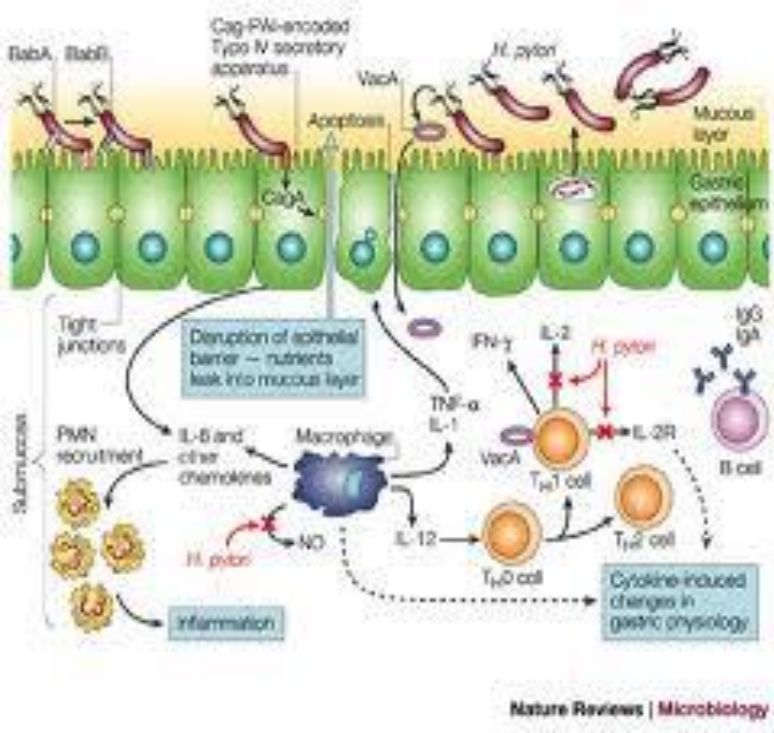
H.pylori has the enzyme urease which splits urea producing ammonia and carbonate.This process creates a environment that facilitates survival of H.pylori in stomach. The ammonia produced in the stomach is damaging to the superficial epithelial cells. There are some mutant strains in the H.pylori which cannot produce urease andwill be unable to colonise the stomach.

H.pylori usually lives in the mucous layer of the superficical epithelial cells.unfortunately the H.pylori strain without flagella are unable to get stirred to the apical membrane of superficial epithelial cells for attachment.these strains are not infective.the mechanism by which it causes injury to stomach maybe through alteration in the acid secretion.this may be due to the fact that inhibitors effect produced by H.pylori upon cells that produce somatostatin,which is a antagonist of antral A cell secreting gastrin .Helicobacter pylori produces decreased

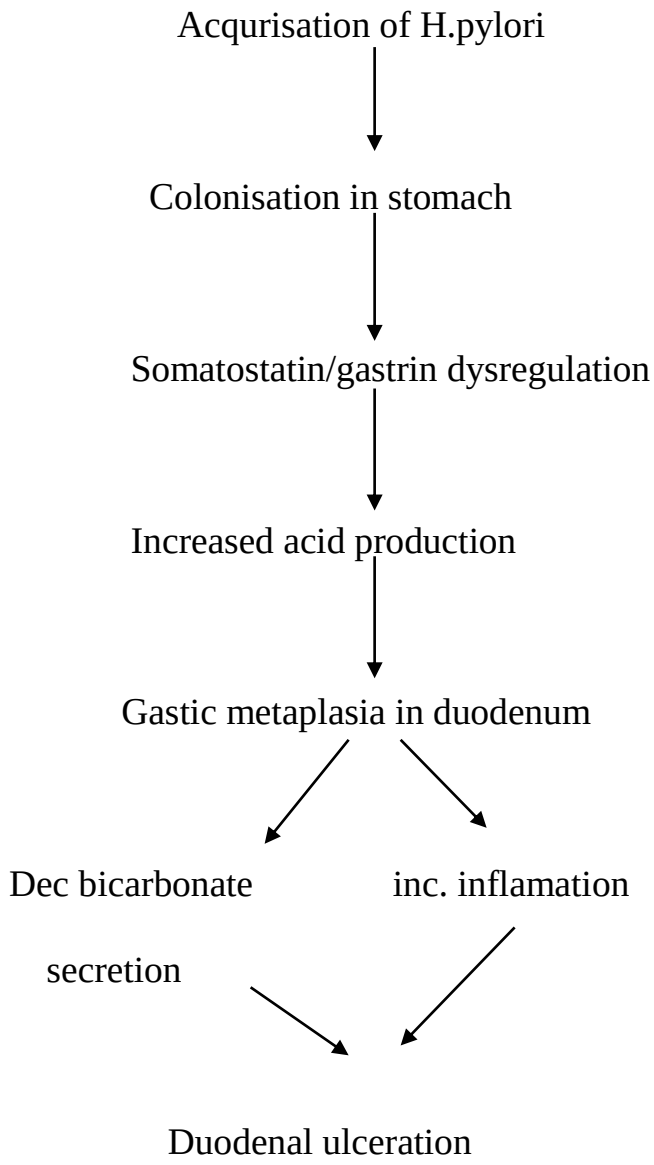
level of somatostatin by decreasing somatostatin messenger rna production and decreased production of less quantity somatostatin producing D cells.

The effects are mainly produced by Helicobacter pylori action of antral alkanisation.Helicobacter pylori activates the release of other local activators and interleukins.the end result in increased production of gastrin and increased production of acid this hypergstrenemia will lead to hyperplasia of parietal cells which is found in many individuals seen who have DU ulcer .

This increased production of acid leads to antral Metaplastic change in the duodenum after the pyloric region. This Metaplastic change makes Helicobacter pylori an environment for colonisation of the duodenum. All patients in this group have increased 50 fold risk of developing duodenal ulcer.H.pylori colonisation produces significant decrease in bicarbonate release from the duodenal mucosa.when H.pylori infection is successfully produced the mucosal mechanism of acid hypersecretion tends to normalise.



Other ways in which H.pylori can produce can induce the injury to gastroduodenal mucosa is through production of toxins namely vac A and cag A.This produces local increased infiltration of cytokines particularly(interleukins) in affected ulcer, infiltration of proinflammatory cells and increase in activation of local immune factors and finally leading the way to apoplasia. The final result is duodenal ulceration.



The evidence support the role of Helicobacter pylori in the production of PUD is very clear. Patients with Helicobacter pylori infection and antral inflammation are 3 times more favourable to

develop the disease when compared to individuals without infecton nearly 92% patients with duodenal ulcer and 70% patients with gastric ulceration have been infected with Helicobacter pylori infection.

It is evident from many of the randomised prospective studies that treatment of pylori infection can produce an alteration in the natural course of PUD. The complete cure of pylori also brings down the repeated attacks of PUD in nearly 75% of individuals on treatment with a course of suppressive acid therapy to nearly < 19% in individuals treated with a course of anti Helicobacter pylori regimen. This clearly states that H.pylori eradication can markedly decrease the incidence of peptic ulcer decrease in the population.

Obviously many other factors also contribute to the production of PUD other than Helicobacter pylori.Every patients who has H.pylori infection do not get peptic ulcer disease.only 10-15% of patients who are colonised with Helicobacter pylori will acquire PUD during their life . Patients who are on aspirin and steroids can develop PUD without Helicobacter pylori infection.These finally conclude that Helicobacterpylori is indoubtfully a critical factor in the formation and repetition of PUD .Helicibacter pylori play a critical role in the production of Stomach carcinoma and lymphomas.

A wide class of abnormalities may be due to exposure of acid to mucosal surface was demonstrated in the individuals with ulcer. As a criteria patients with ulcers in duodenum tend to produce increased acid secretion when compared with individuals having stomach ulcer. It was noted that ulcer in duodenum individuals have been a increased mean basal acid output than normal levels.

Nocturnal secretion of acid is increased when compared with Secretion during morning hours .However most of the persons show their basal and peak outputs under normal limits and there is no relation to acid secretion and nature of the disease. Patients with duodenal ulcer produce increased secretion output to a secretary stimulus than patients with normal controls .Even though duodenal ulcer patients usually have falling gastric levels normal. They can produce enormous amount of gastric acid in response to gastrin than normal individuals.

Considering that patients with duodenal ulcer can produce excessive gastric acid.it has been said that that there are individuals have high falling gastrin levels.there is a decreased feedback mechanism in view that the parietal cell mass have increased sensitivity to gastrin.many of the long standing randomised trials have reasonably given a good understanding of acid and gastrin production in patients affected with

H.pylori infection.some individuals who have duodenal ulcer have an increased rate of gastric emptying which deliver an increased acid output to the duodenum.finallhy the buffering action of duodenal mucosa is diminished due to decreased production of bicarbonate secretion in individuals with peptic ulcer disease.

In individuals who present with gastric ulcer the acid secretion is quite variable.currently stomach ulcers are divided according to the Modified Johnson classification.

Type I-located near angularis incisuraris lesser curvature.

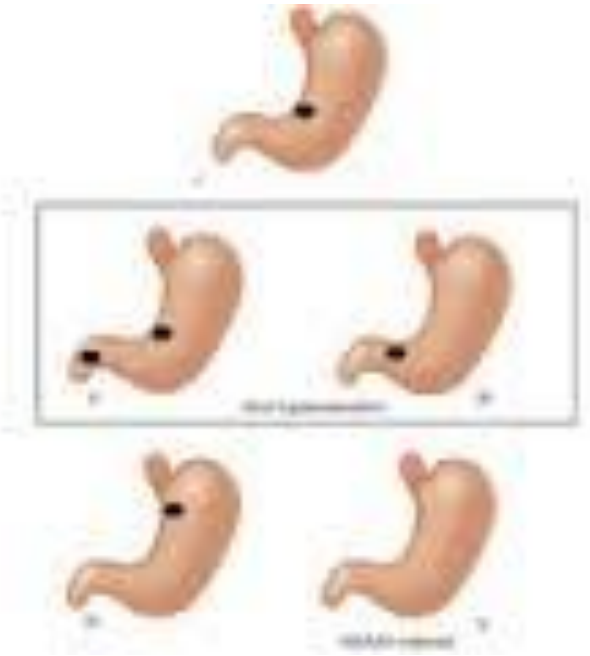
Type II-associated with active or quiesent duodenal ulcer

Type III-prepyloric disease

Type IV-near gastroesophageal junction

Type V-medication induced can occur anywhere in stomach

MODIFIED JOHNSON CLASSIFICATION



Individuals with gastric ulcer have decreased mucosal defences that lead to abnormal amount of acid back entry into mucosa producing injury.Duodenogastric reflex will lead to weaking of gastric mucosal defences.

GIANT DUODENAL ULCERS

These are a group of ulcers that might produce heavy morbidity in individuals. It is defined as a complete thickness defect that is 2 cm in length or greater in diameter and involves a greater portion of duodenum. These ulcers were first described by BRITZKA in 1931 that involve a greater part of duodenum

Giant duodenal ulcers contribute to 1% of all duodenal ulcers and 5% of all peptic ulcer disease which require emergency surgical interference. The ratio of male and female being affected is 3: 1.

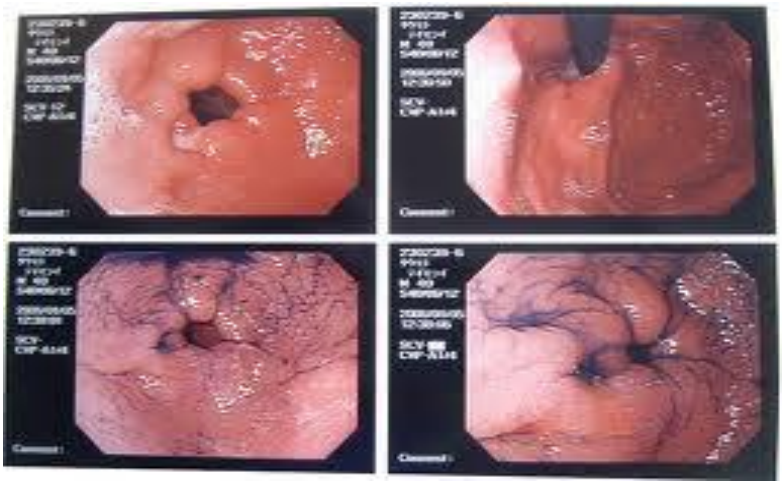
The main reason for giant duodenal ulcers are now recognised to be as the usage of Nsaids rather than h pylori. From the previous studies it has been clear that highest percentage of cases of giant duodenal ulcer is due to NSAIDS.

Perforation due to giant duodenal ulcer is very critical due to excessive amount of destruction of duodenal tissue and a overwhelming sepsis occurs.

Giant duodenal ulcers have a higher chance of post operative leak syndromes and gastric outlet obstruction.

ENDOSCOPICAL VIEW OF GASTRIC AND DUODENAL

ULCERS



MACROSCOPIC VIEW OF GASTRIC ULCER WITH EVERTED

Edges of ulcer shows everted margins with a central crater

NSAIDs in peptic ulcer disease

NSAIDs are inevitably linked to peptic ulcer disease .Individuals who are on treatment for rheumatoid arthritis and osteoarthritis take NSAIDs.these patients are affected to 10- 15% with PUD . The prevalence of peptic Ulcer disease among persons who use NSAID is about 24%. These involve 15% mainly gastric ulcer and 10% include duodenal disease PUD complaints such as perforation and bleeding are more common in NSAID users. most of the patients are brought into focus only after facing the complications the risk of getting complications and adverse effects in patients using NSAID is higher when compared to controls.The risk is higher and older age group.

In old patients who are under treatment using NSAIDs are likely to acquire an surgery relating to GIT problem is ten times when compared to normal subjects. It was also found that the mortality rate in these individuals have increased to higher limit than the normal persons. They also have a higher incidence of hospitalisation rate for gastric intestinal problems than the normal subjects.

Recently the increased usage of NSAID like aspirin has increased due to increase in cardiovascular accidents in the elderly.so these

patients must receive a concomitant acid suppression therapy if any of the risk factors are found.

Risk factors for usage of concomitant acid suppressive therapy

1. ager over 80
2. history of acid peptic disease
3. concurrent steroid intake
4. concurrent anticoagulant intake
5. high dose NSAID

Other factors

A large number of epidemiological studies prove that persons who smoke are about two times more prone to develop PUD than non smokers.Smoking increases acid output and increases duodenumgastric reflex.Smoking increases the amount of acid secretion and decreases prostaglandin secretion and bicarbonate secretion. Many studies have proven the relation of smoking to peptic ulcer disease.Although difficult to measure plays both physical and psychological stress plays an important role in the treatment of peptic ulcer disease.

In 1842 cushing decribed duodenal ulcer and duodenitis in burns patients later he explained the duodenal ulceration in the head injury patients later it was named as Cushing's disease.

Clinical manifestations

More than 90% of the individuals with peptic ulcer disease complain to have abdominal pain the pain is typically burning in character and usually localised to epigastrium the real cause of pain is unclear the character of the pain is usually two to three hours after having a meal. Majority of the patients will have a severe pain which will make them to awake from sleep. Gastric ulceration pain usually occurs during food intake and the pain usually subsides after that. It does not awake the patient from sleep a careful history about peptic ulcer disease, use of steroids and other anti secretary medication is indicator of the diagnosis.

Signs and symptoms

-nausea

-bloating

-loss of weight

-stool positive for occult blood

-even anaemia

-melena

Duodenal ulcer usually occurs in male when compared to females gastric ulcer on the other hand do not have such a sex prediction and have a equal incidence in male and female patients.

Diagnosis

In young individuals who complain of having dyspepsia or epigastric pain, it is enough to start an empirical dose of protein pump inhibitors to treat peptic ulcer disease without doing a confirmatory test in these case it is always necessary to inform the patients that there may be a little possibility of malignant lesion even of the symptoms of ulcer disease decrease markedly. In old individuals above 45 years of age must be subjected to an upper GI endoscopy all patients who have warning symptoms must undergo GI endoscopy irrespective of age.

Alarming symptoms

1.weight loss

2.recurrent vomiting

3.dysphagia

4.bleeding

5.anaemia

Investigation for peptic ulcer disease

- upper GI endoscopy
- biopsy
- a double contrast upper GI Xray
- any site of gastritis must be biopsised to rule out infection by Helicobacter pylori
- bottom line serum gastrin level to find out the possibility of gastrinoma

Indications for diagnosis and treatment of H.pylori

Established

Active peptic ulcer disease

Previous history of PUD not being treated for H.pylori

Low grade gastric mucosa associated lymphoid tissue

After the resection of early gastric carcinoma by endoscopy

Controversial

Non ulcer dyspepsia

GERD

Persons using steroids

Unexplained anaemia

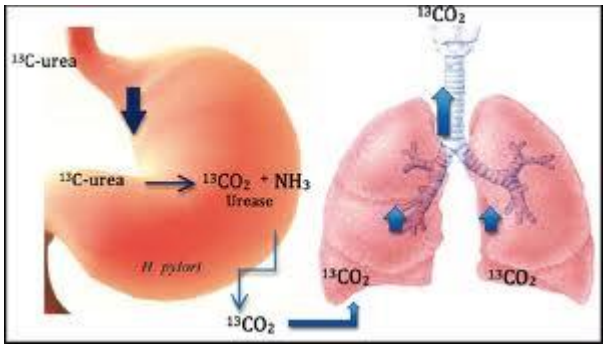
Population at high risk for gastric carcinoma

Tests for Helicobacter pylori

H.pylori has been known to produce peptic ulcer disease and has a role in gastric lymphoma and adenocarcinoma.there are a number of test to detect the presence of H.pylori.the positive predictive value of the test used for screening the H.pylori usually depends upon the prevalence of the H.pylori infection in the population to be investigated.

A positive test is usually holds good in saying the H.pylori infection in the locality but the negative predictive test is usually unreliable so in a clinical setting treatment for H.pylori infection must be initiated on the basis of positive predictive value.

Due to strong association of H.pylori infection with gastric lymphoma and carcinoma many clinicians have recommended treating of H.pylori infection when their diagnosis is made.



-a positive serological test is a direct evidence of active infection

-histological examination of antral mucosal biopsy using special strains is the standard test

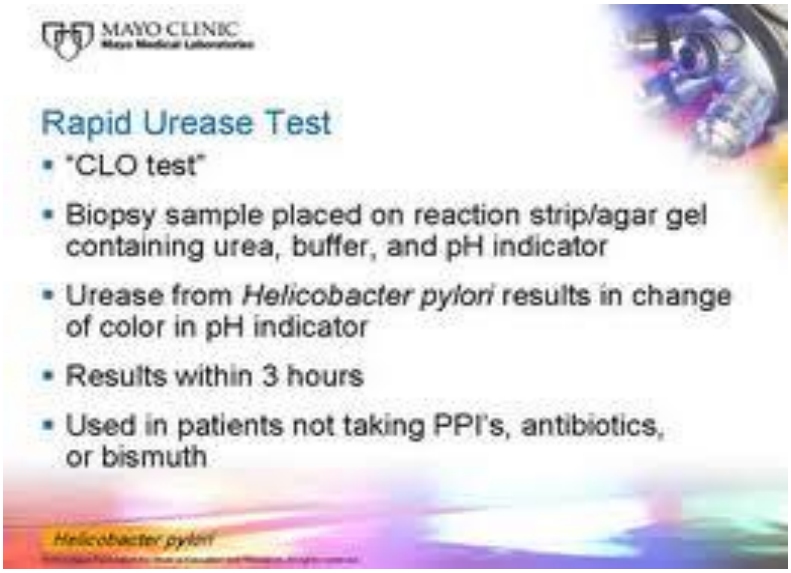
-RAPID UREASE TEST-to detect the presence of urease in mucosal biopsies

Urease test is a strong evidence of infection with H.pylori.urease is a common enzyme that is found abundant in H.pylori strains.this urease enzyme is necessary for colonisation of H.pylori in the gastric mucosa.

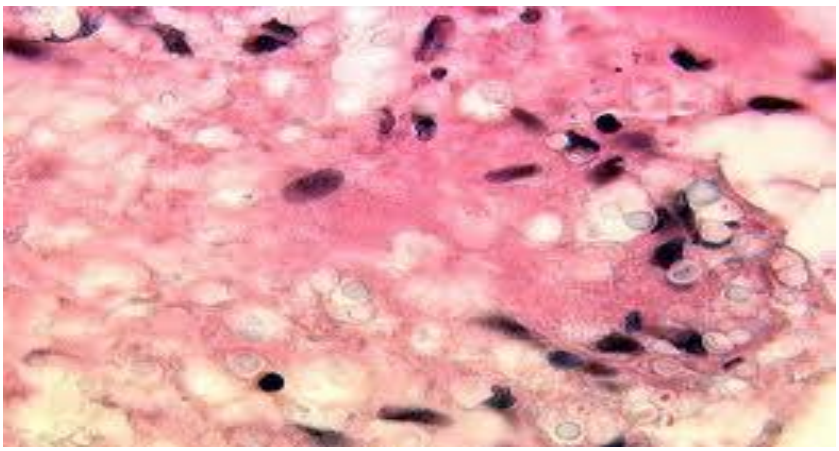
The labelled carbon-13 urea breath test has been developed as a reasonable test to register the eradication of H.pylori after a considerable treatment is given.

In this test urea is taken in food labelled with nonradioactive carbon- 13.This urea labelled is broken down by the urease produced in enormous amount by the H.pylori and they are divided into ammonia and carbon di oxide.the carbon di oxide radiolabelled is expired via the lungs and can be detected in the air expired.Blood sample can also be taken to detect the presence of radiolabelled carbondioxide Another test that is used is fecal antigen test which is quite sensitive and specific for a overwhelming H.pylori infection

Rapid urease test kit



The biopsy specimen is kept on the strip that contains urea,buffer and ph indicator.Since hpylori contains excessive amount of urease it produces change in color in the ph indicator . The usefulness of the technique sis that it can be read within 3 hours.



Histopathological examination of staining with urease kit

Complications of the peptic ulcer disease

-bleeding

-perforation

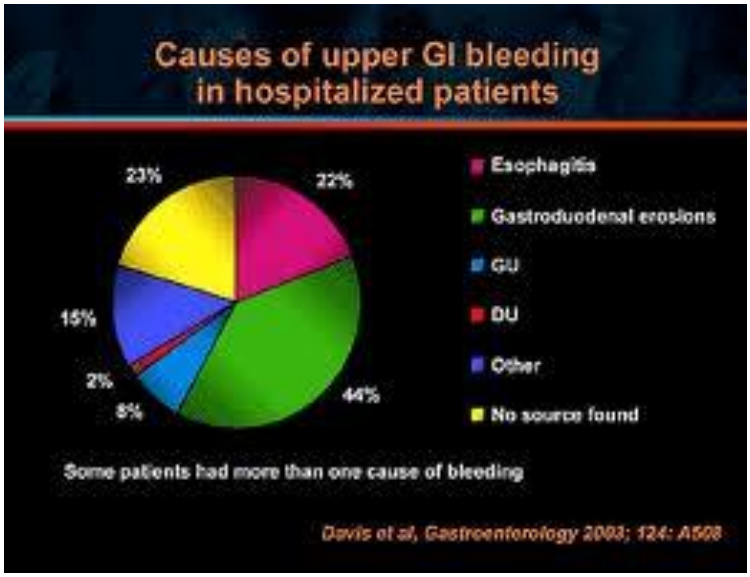
-obstruction

Causes of upper GI bleeding

Peptic ulcer disease is the most common cause for the upper GI Bleeding Patients who have upper GI bleeding must be admitted to hospital and resuscitated patients with bleeding ulcers have symptoms of melena and haematemesis.

This can be confirmed by nasogastric aspiration.abdominal pain can be a presenting symptom .Profound blood loss patients may require a aggressive resuscitation and blood transfusion.

The best form of treatment is to diagnose and identify the cause of bleeding and arresting them .Nearly 75% patients admitted in hospital with PUD bleeding will recover if they are supplemented with acid suppressive therapy. However remainder of patients have a tendency to rebleed.



The patients who have the tendency to rebleed depends upon the magnitude of the bleeding,age and endoscopic findings.

High risk group patients

-shocks

-haemetemesis

-transfusion necessary for 4 units in 24 hrs

-certain endoscopic stigmata

continuous bleeding

Visualised vessel

Increased risk patients are usually benefitted from endoscopical therapy to control active bleeding. The commonly used modalities are injection using epinephrine.

There are two important widely used risk stratification tools which has been found useful in predicting rebleeding and death

Blantchford score

Rockall score

Blatchford score

At presentation			score
1.systolic BP			
100-109mm Hg			1
90-99mm Hg			2
<90mm Hg			3
2. blood urea nitrogen			
6.5-7.9			2
8.0-9.9			3
10-24.9			4
>25			6
Hb	men	women	
	12-12.9	10-11.9	1
	<10	<10	6

Other variables

Pulse >100 beats/min	1
Melena	1
Syncope	2
Hepatic disease	2
Cardiac failure	2

Rockall score

Clinical	score
Age	
<60yrs	0
60-79yrs	1
>80yrs	2
Shock	
Heart rate>100	1
Systolic BP<100mm Hg	2

coexisting illness	
ischemic heart disease, congestive cardiac failure	2
renal, hepatic failure metastatic cancer	3
endoscopic diagnosis	
no lesions observed,mallory weiss	0
Peptic ulcer,erosive disease	1
cancer of upper GIT	2
endoscopic stigmata	
clean base ulcer,flat pigmented spot	0
blood in upper GIT active bleeding	2

ENDOSCOPIC TREATMENT FOR BLEEDING:

Bleeding ulcers can be treated endoscopically by a different number of ways. Nowadays the options has increased dramatically.

Using endoscopic clips

Heater probe for coagulation of lumen

Epinephrine injected at the base

For diffuse areas of bleeding argon beam laser

Bicap probe

Endoscopic arrest of ulcer bleeding requires various skill and good training in this field . The main difficulty in this technique is that it is very tough to see the bleeding vessel through the scope when there is active bleeding.

If the endoscopist fail in controlling the bleeding Process or the patient rebleeds it is better to shift the patient to Radiology or surgical department for further evaluation. If however the patient has been stabilised hemodynamically and again if he bleeds further another

endoscopy can be done to control the bleeding if no other large vessel is found to be bleeding which can detoriate the status of the individual.

Perforation

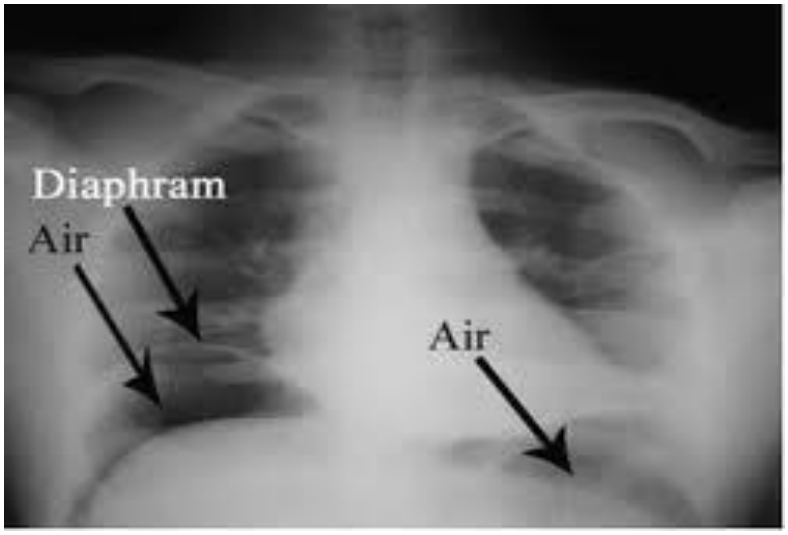
Perforated peptic ulcer usually present to the hospital as an abdominal emergency.The individual usually gives the absolute period of start of unbearable pain.

Two phase

-chemical peritonitis; due to gastric and duodenal secretion

-bacterial peritonitis

As a result there is fluid sequestration in the third space resulting in dehydration and this would make recorrection using fluids a mandatory situation.the patient would have symptoms of abdominal pain and a very obvious distress can be seen



Air under diaprahgm

Peritoneal signs of perforation

-guarding

-rebound tenderness

-air under diaphragm 80% may be seen

Once the diagnosis is made it may be resusicated with normal saline and after sterilisation can be taken for surgery.Sometimes the perforation can get sealed by itself and surgery can be avoided.the sealed perforation can be confirmed by a radiological contrast study.

Perforated duodenal ulcer actually manifest as sudden increase in epigastric pain which may continue to affect the entire abdomen

cavity.usually the pain is caused by sudden gush of gastric contents into the peritoneal cavity

This pain usually reaches a high intensity suddenly and then Afterwards become a continuous pain which is aching in character with restriction of abdominal movements.

There will be pain radiation to the right scapular region due to the fact that the released gastric contents occupy region of right sub phrenic space. Peritoneal contamination with irritation becomes very high and most patients will stay still to decrease their movements.The factors affecting the abdominal findings include

size of defect in gastric mucosa

total content of organisms and secretions in peritoneum

interval between onset and time during admission

spontaneous closure

PHYSICAL EXAMINATION

Fever

Decreased intestinal peristalsis

Rigidity in anterior abdominal wall

There may be symptoms of

Shock

Tachycardia

Decreased output in urine

Hypotension

The other indicators of toxicity may not be found in patients who are Severely immunocompromised and older individuals. The differential Diagnosis of perforation include

Acute MI

Aortic dissection

Cholecystitis

Pancreatitis

Appendicitis

Renal colic

IMAGING MODALITIES

Chest xray pa view or a lateral decubitus xray to demonstrate free air which will be found in nearly 70 to 80% of individuals. Air under diaphragm is not only found in duodenal or gastric ulcers it is also found in other hollow viscus perforation .Pneumoperitoneum is present in larger amounts in a case of duodenal or gastric ulcers than other abdominal viscera.

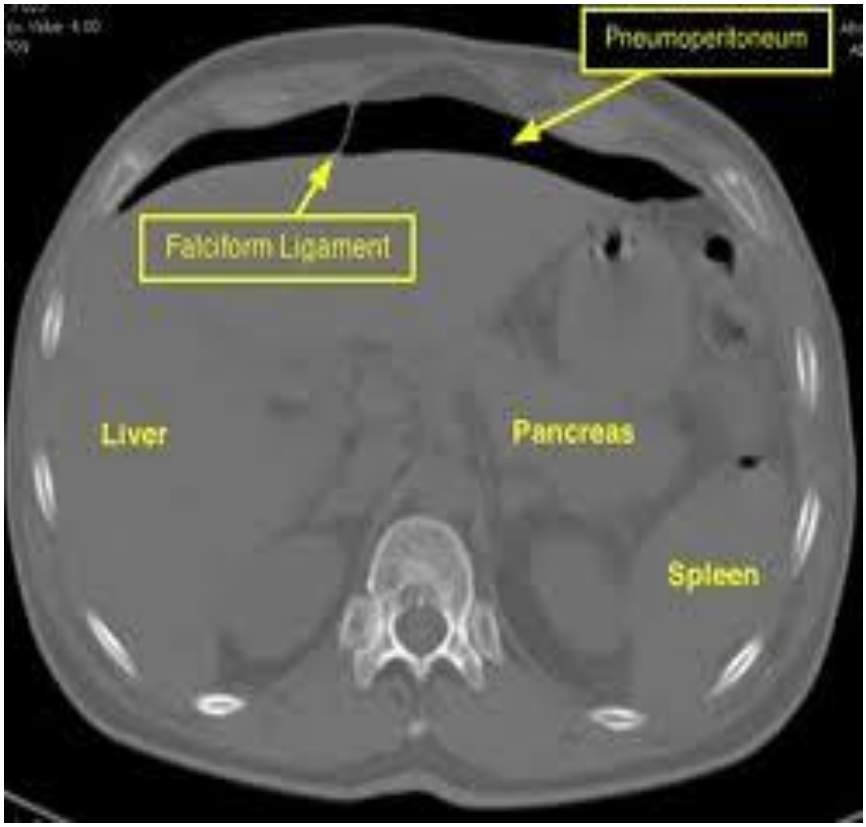
Upper gastrointestinal study with water soluble contrast using

Gastrograffin which will demonstrate release of contrast through the defect

Gastograffin contrast leak in xray

Plain ct abdomen will show presence of free air in peritoneal cavity.

CT with contrast may show leak of contrast from perforated site



CT showing air in peritoneal cavity : pneumoperitoneum

RISK STRATIFICATION:

The overall risk factors contributing to the prognosis usually includes

Start of treatment after 24 hours

Hypotensive shock before surgery

Comorbid conditions

TREATMENT

Graham omental patch repair is the most commonly performed Procedure

Some patients may need further additional procedures in certain circumstances

Perforations usually greater than 2.0 cm may need THAL patch or they may require vagotomy,bill roth procedure and antrectomy Synchronous bleeding and perforation may need resection pyloroplasty,u stich control

Chronic ulcer symptoms :patch closure with parietal cell vagotomy or pyloroplasty with ulcer excision Nsaids dependence : patch closure , parietal cell vagotomy, pyloroplasy or ulcer excision

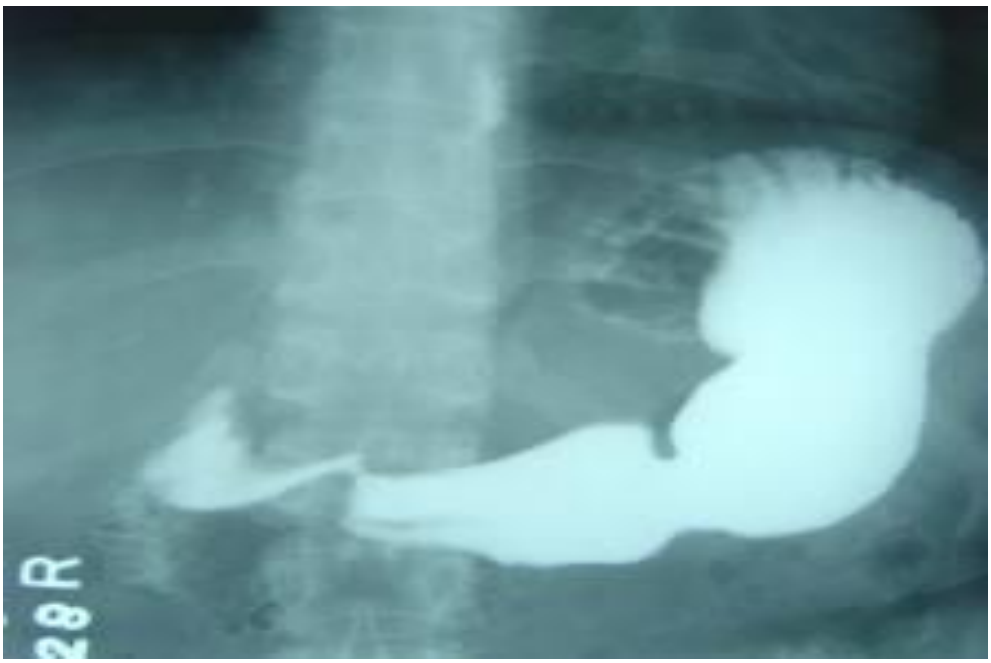
Previous h pylori treatment failure :patch closure, parietal cell vagotomy,pyloroplasty and ulcer excision

Previous ulcer complications :patch closure , parietal cell vagotomy pyloroplasty,and ulcer excision

Obstruction

Usually occur in about less than 5% of patients it may be usually due to a duodenal or pre pyloric disease. The obstruction can develop due to inflammatory lesion and peristaltic dysfunction or may be for longer periods due to ulcer formation

barium meal series showing distended stomach and accumulation of contrast within the stomach and deformed duodenal cap



CT ABDOMEN SHOWING GASTRIC OUTLET OBSTUCTION

Signs and symptoms

1.non bilious vomiting

2.abdominal pain

3.profound hypokalemic hypocholermic metabolic alkalosis

4.weight loss

Treatment

1.nasogastric suction

2.IV hydration

3.anti secretory medication

4.diagnosis confirmed by endoscopy

5.May require a balloon dilation or surgery

6.malignancy has to be ruled out.

Medical Management

‘Proton pump inhibitors are the main stay of treatment in case of PUD.patients who are hospitalised for ulcer complication must be given with a IV infusion of proton pump inhibitors these individuals are candidates for lifelong PPI. peptic ulcer patients should quit alcohol and smoking and NSAID use.

Patients who definitely require a NSAID therapy must be given with addition of PPI.If H.pylori is found to be present it has to be treated adequately.

Usually anti secretory drugs can be withdrawn after three months.if the causative agents such as H.pylori,NSAID or aspirin is removed misoprostol,sucralfate and acid suppression therapy can be accepted.

Treatment regimens for H.pylori

Medication	duration
PPI+clarithromycin 500mg+amoxicillin	10-14 d
PPI+clarithromycin+mebonidazole	10-14d
PPI+amoxicillin,then	5 days
PPI+clarithromycin+tinidazole	5 days

Other regimens

Bismuth salicylate+metronidazole+tetracycline+PPI	10-14 days
PPI+amoxicillin+levoflox	10 days

Surgical management

The indications of the surgery in case of PUD is bleeding, perforation and obstruction and non healing ulcer.stomach carcinoma must be kept in mind when treating patients with gastric ulcer or outlet obstruction.

Common procedures usually done is

Simple oversewing of a bleeding ulcer

Omental patch to a perforated ulcer

Distal gastrectomy

During the past decades most of the peptic ulcers was Satisfactorily treated with

Parietal cell vagotomy

Vagotomy and drainage

Vagotomy and distal gastrectomy

Parietal cell vagotomy

This is a safe procedure and saves nerve supply to upper one third of stomach in which the parietal cells are localised.this does not involve the vagal supply to antrum and pylorus.this is the way the production of acid is decreased by 75%HSV is very useful in case of gastric ulcer except in type II and type III ulcers because of hypergastrenemia caused due to outlet obstruction and stasis in antrum.the Taylor procedure

consists of a posterior truncal vagotomy and includes anterior seromyotomy.

Vagotomy and drainage procedures

This includes truncal vagotomy and pyloroplasty and Gastrorejejunostomy. The greatest facility of V+D procedure is that it can be performed in a fast and safety manner .Disadvantages include significant dumping and diarrhoea the main complication of this procedure to be kept in mind is the perforation of esophagus.

It is useful in

1.bleeding duodenal and gastric ulcer

2.perforated duodenal and gastric ulcer

3.obstructive duodenal and gastric ulcer(type II and III)

The drainage procedures usually done in case of V+D procedures are

Gastrojejunostomy

Pyloroplasty

Commonest pyloroplasty technique used is Heineke-

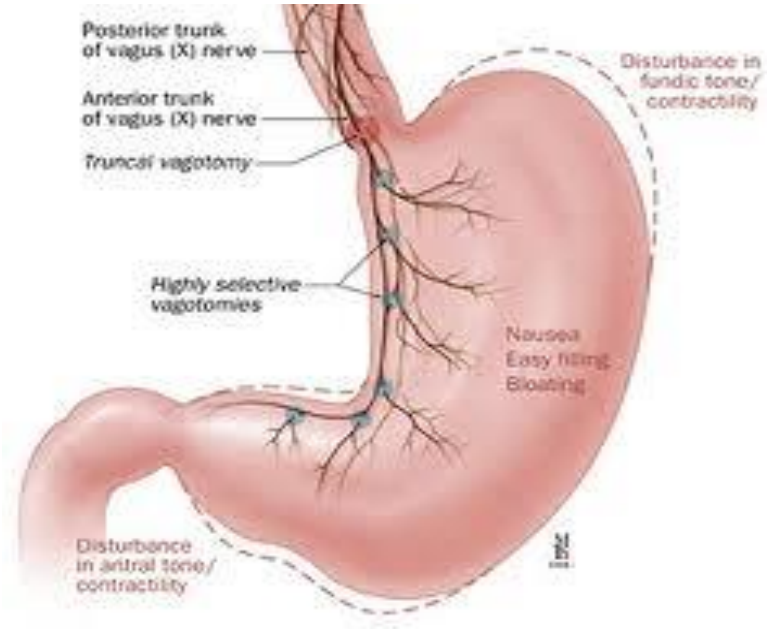
Mikulicz type other types which are rarely used is Jaboulay pyloroplasty

Vagotomy and antrectomy

This technique has a very low recurrence rate and is applicable to all patients with peptic ulcer disease the ulcer is usually included in the specimen removed and remaining is established by Billroth I and II gastrojejunostomy.Roux-en-y procedure is usually avoided since this procedures leaves a stomach remnant of about 60-70%.this procedure is usually done as a elective procedure and its role in emergency is questionable and cannot be done in patients with extensive inflammation.

Surgical options in treatment of duodenal and gastric ulcer

Indication	**Duodenal**	**Gastric**
Bleeding	1.oversew	1.oversew biopsy
	2.oversew,V+D	2.V+D
	3.V+A	3.distal gastrectomy
Perforation	1.patch	1.biopsy,patch
	2.patch,HSV	2.wedge excision,V+D
	3.patch V+D	3.distal gastrectomy
Obstruction	1.HSV+GJ	1.biopsy,HSV+GJ
	2.V+A	2.distal gastrectomy
Non healing	1.HSV	1.HSV and wedge excision
	2.V+D	2.distal gastrectomy



The picture shows the anterior and posterior branch of vagus nerve and site where truncal vagotomy is done and site where highly selective vagotomy is done

ENDOSCOPIC VIEW OF DUODENAL ULCER DURING OGD

ULCER IS SEEN IN THE FIRST PART OF DUODENUM.

MATERIALS AND METHODS

The study involves the patients who present to the department of general surgery with symptoms of dyspepsia subjecting to endoscopy. Among these patients who are subjected to endoscopy ,those who are having duodenal ulcer are taken into study.

After documentation of history, clinical examination involves general physical examination is done.During endoscopy the stomach wall is examined and any changes in stomach wall is noted .Endoscopically and biopsy from two areas in stomach is taken from antrum and body and sent to histopathological examination.

During histopathological examination the specimen is subjected to rapid urease test to confirm the presence of H.pylori.the histopathological changes are noted and the lesions are recorded.

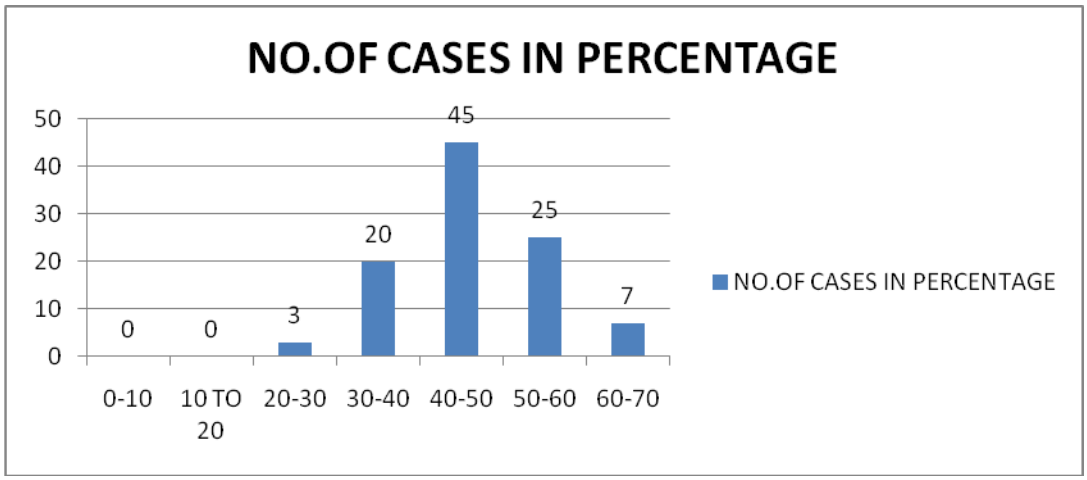
Based on this study we analyze the incidence of different type of lesions such as chronic atropic gastritis,superficial pan gastritis in these patients other than site of peptic ulcer disease and their association with H.pylori.

Exclusion criteria: carcinoma stomach ,gastric polyps, alcoholic gastritis.

RESULTS AND OBSERVATION

AGE RELATED

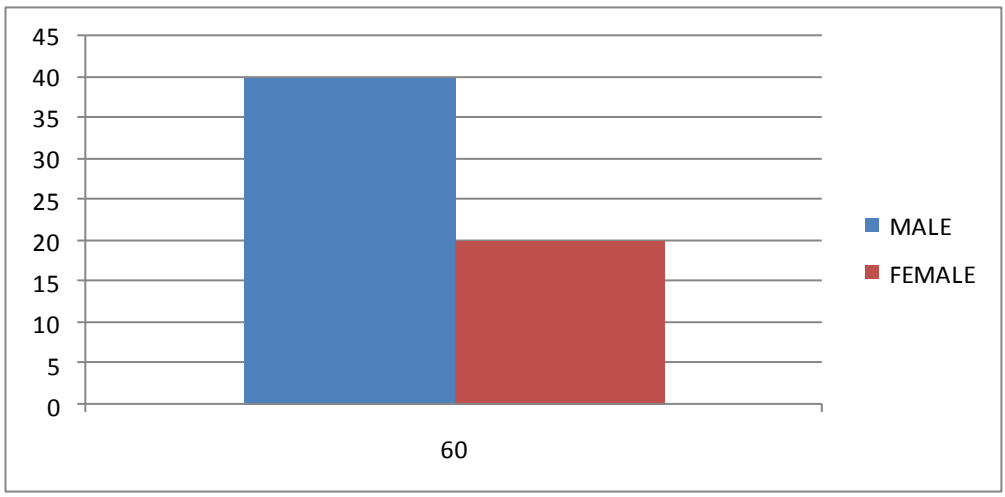
AGE	%
0-10	0
10-20	0
20-30	3
30-40	20
40-50	45
50-60	25
>60	7



Here we are able to find that incidence of cases is nearly 75 % in the age group between 40 to 50 years and duodenal ulcers are uncommon below 20 years of age

SEX RELATED

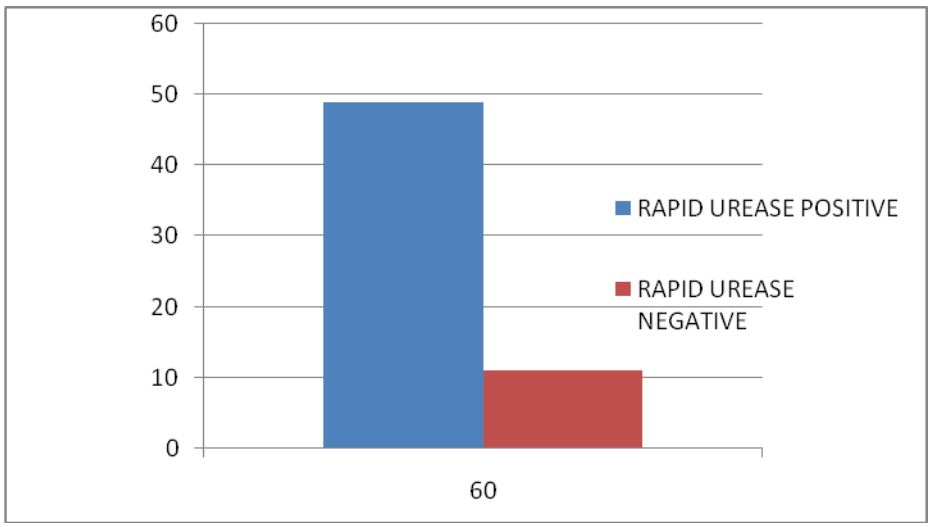
NO.OF CASES	MALE	FEMALE
60	40	20



The percentage of cases affecting male was nearly 66.6% and in the female population it is nearly 33.3%.Hence males are more prone to get peptic ulcer disease than woman

ASSOCIATION OF H.PYLORI

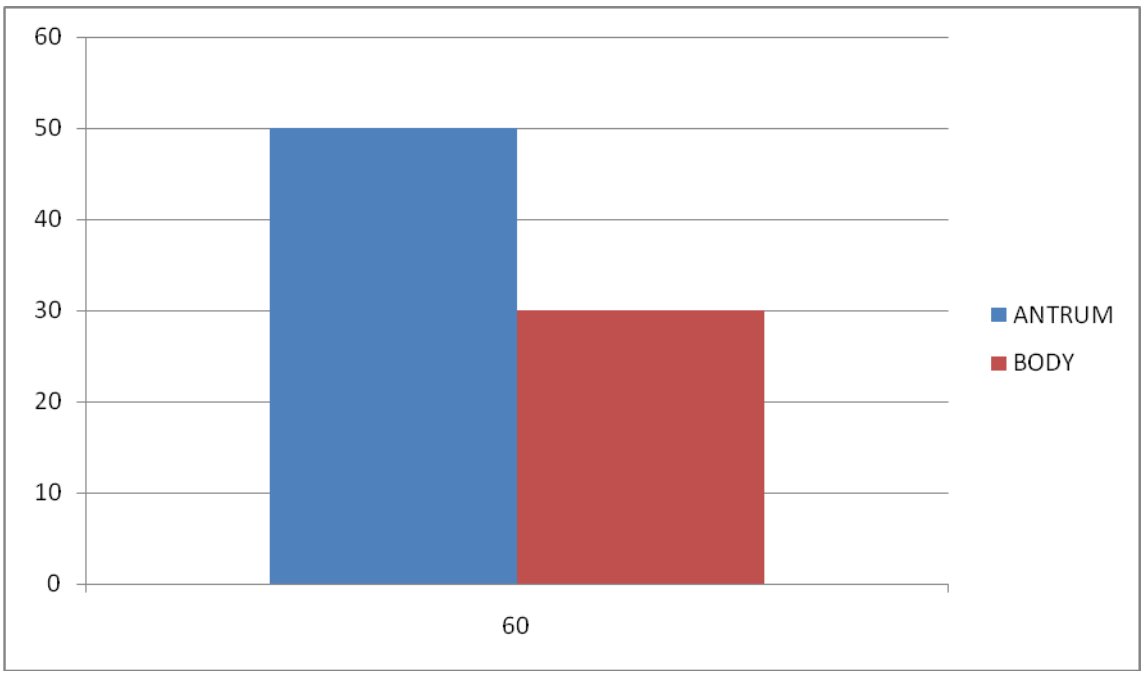
NO.OF CASES	RAPID UREASE POSITIVE	RAPID UREASE NEGATIVE
60	49	11



The percentage of cases which were urease positive in a case of duodenal ulcer was nearly 87.6% and urease negative was 12.4%. Hence patients with duodenal ulcer have been mostly colonised with h.pylori.

COMMONEST AREA TO BE AFFECTED

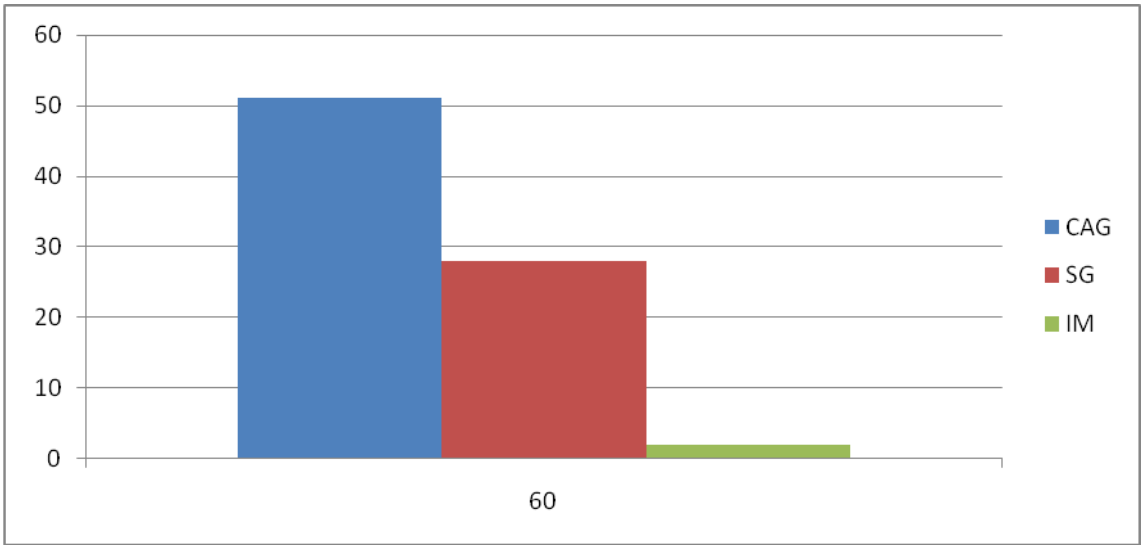
NO.OF CASES	ANTRUM	BODY
60	50	30



The percentage of cases in which antrum was affected was nearly 84% and 54% in body of stomach was affected by h pylori in nearly 50% of cases hence we found that antrum is the commonest site affected by hpylori

TYPE OF LESION

NO.OF CASES	CAG	SG	IM
60	51	28	2



CAG-CHRONIC ATROPIC GASTRITIS

SG-SUPERFICICAL GASTRITIS

IM-INTESTINAL METAPLASIA

The overall incidence of chronic atropic gastritis is nearly 84.1% when compared to other type of lesions. Hence it is the most commonest lesion caused by h.pylori.

DISCUSSION

The study tells us about the incidence of H.pylori infection in patients having chronic duodenal ulcer and the different types of lesions found endoscopically and proved histopathologically. Of the 60 patients under study 48 patients were found to be positive for H.pylori using rapid urease test positive in antrial biopsy.20 patients were found to have been H.pylori positive in specimen with biopsy from the body of the stomach.

The histopathological findings on these were usually atropic gastritis, superficial pangastritis and intestinal metaplasia. In antrum the commonest histopathological change was chronic atropic gastritis nearly 80% and the remaining showed 18.5%patients have superficial gastritis including the mucosa and submucosa.nearly 1.5% patients showed the features of intestinal metaplasia.

Moreover patients were mainly males nearly 80%and females were 20% affected due to H.pylori.the old individuals are mostly affected than the younger population.the prevalence rate of H.pylori infection in colonisation of stomach wall in antrum was nearly 92% and the body showed colonisation in 46% of cases.

As a comparison to study conducted by Dr jagmohan , in his study there were totally 100 patients in his study of which there 74% males and 24% females affected .In our study percentage of males affected were 66% and females were 33%.

The histological findings in that study was chronic gastritis of antrum was 93% and fundus was affected in 66% of cases.in our study the percentage is 92% and 42% respectively.

The overall incidence percentage of chronic atropic gastritis of in that study was 82% and percentage of cases in our study is 84%.

Hence in both studies compared the percentage values correlate each other.

CONCLUSION

Finally after conclusion from the study and comparing with previous studies we are able to get the following results,

the prevalence of H.pylori infection is more common in antrum than body of stomach

the commonest histopathological change in the stomach wall produced by H.pylori is chronic gastritis which is of atropic type

men are usually affected than women by H.pylori

the elder population has higher incidence than the younger population.

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PROFORMA

CHANGES IN STOMACH WALL OTHER THAN SITES OF ULCER IN PEPTIC ULCER DISEASE.

Patient details patient ID NO:

Name:

Age/sex:

IP NO:

DOA:

DOE:

Address:

History

Occupation:

Socioeconomic status:upper/upper middle/lower middle/poor

ABDOMINAL PAIN:- duration:- onset:-

VOMITTING:- duration:- onset:-

INDIGESTION:-

H/O DRUG INTAKE/ALCOHOLISM

Family history:

Past history:DM/HT/Asthma/TB/Other(surgery)

General examination

CONSIOUS

ORIENTED

AFEBRILE

ICTERUS

ANEMIC

PR

BP

Examination of abdomen

EPIGASTRIC TENDERNESS:PRESENT/ABSENT

MASS :PRESENT/ABSENT

Investigations

Hb:

TC:

DC:P L E M

ESR:

Blood sugar:

Blood urea:Serum creatinine:

ENDOSCOPY:

HISTOPATHOLOGICAL REPORT:

ANTRUM:

BODY:

RAPID UREASE TEST:POSITIVE/NEGATIVE

MASTER CHART

sl no	NAME	IP NO	AGE	SEX	SYMPTOMS	ENDOSCOPIC FINDINGS		HISTOPATHOLOGICAL FINDING		RAPID UREASE TEST	
						ANTRUM	BODY	ANTRUM	BODY	ANTRUM	BODY
1	loganathan	17840	72	m	dyspepsia	gastritis	gastritis	AG	SG	P	P
2	rajeswari	18755	45	f	abdominal pain	gastritis	normal	AG	NOMAL	P	N
3	SARAVAN	19131	40	M	dyspepsia	gastritis	normal	AG	NOMAL	P	N
4	selvam	19084	42	m	abdominal pain	gastritis	gastritis	AG	AG	P	P
5	suresh	18891	28	m	epigastric pain	normal	normal	NORMAL	NORMAL	N	N
6	marimuthu	19001	46	M	epigastric pain	gastritis	gastritis	AG	SG	P	P
7	sundarraj	18686	55	m	dyspepsia	gastritis	normal	SG	NORMAL	P	N
8	ravi	19248	45	m	epigastric pain	gastritis	normal	SG	NORMAL	P	N
9	sriman	19246	46	m	dyspepsia	gastritis	gastritis	AG	AG	P	P
10	siva	19254	46	m	abdominal pain	gastritis	normal	SG	NORMAL	P	N
11	rani	18908	45	f	dyspepsia	gastritis	gastritis	AG	SG	P	P
12	shamth b	18996	45	f	dyspepsia	gastritis	normal	AG	NOMAL	P	N
13	siva	19328	26	m	epigastric pain	gastritis	normal	AG	NOMAL	P	N
14	ezhumalai	19252	40	m	dyspepsia	gastritis	gastritis	AG	AG	P	P
15	sekar	16179	45	m	epigastric pain	normal	normal	NORMAL	NORMAL	N	N
16	natarajan	18136	48	m	dyspepsia	gastritis	gastritis	AG	SG	P	P
17	palani	19253	70	m	abdominal pain	gastritis	normal	IM	NORMAL	P	N
18	viji	19488	40	f	dyspepsia	gastritis	normal	SG	NORMAL	P	N
19	vinoth kumar	19489	53	m	abdominal pain	gastritis	gastritis	AG	AG	P	P
20	bhavan das	19521	71	m	epigastric pain	gastritis	normal	SG	NORMAL	P	N
21	ganesh raja	19856	38	m	epigastric pain	gastritis	gastritis	AG	SG	P	P
22	sampath kumar	19816	42	m	dyspepsia	gastritis	normal	AG	NOMAL	P	N
23	hussain	20509	34	m	epigastric pain	gastritis	normal	AG	NOMAL	P	N
24	joseph	20501	50	m	dyspepsia	gastritis	gastritis	AG	AG	P	P
25	sivalingam	20517	38	m	abdominal pain	normal	normal	NORMAL	NORMAL	N	N
26	valliammal	19574	48	f	dyspepsia	gastritis	gastritis	AG	SG	P	P
27	govindsamy	20521	75	m	dyspepsia	gastritis	normal	SG	NORMAL	P	N
28	ravikumar	20018	45	m	epigastric pain	gastritis	normal	SG	NORMAL	P	N
29	venkatasamy	19486	60	m	dyspepsia	gastritis	gastritis	AG	AG	P	P
30	mutthaia	19840	62	m	epigastric pain	gastritis	normal	SG	NORMAL	P	N
31	balu	19700	57	m	dyspepsia	gastritis	gastritis	AG	SG	P	P
32	shanta	19828	38	f	abdominal pain	gastritis	normal	AG	NOMAL	P	N
33	jagannathan	19076	60	m	dyspepsia	gastritis	normal	AG	NOMAL	P	N

34	koteswari	20017	48	f	abdominal pain	gastritis	gastritis	AG	AG	P	P
35	gurumoli	19988	60	f	epigastric pain	normal	normal	NORMAL	NORMAL	N	N
36	parthasarathi	20809	47	m	epigastric pain	gastritis	gastritis	AG	SG	P	P
37	neelavathi	20881	45	f	dyspepsia	gastritis	normal	SG	NORMAL	P	N
38	chinathambi	20011	66	m	epigastric pain	gastritis	normal	SG	NORMAL	P	N
39	mahalingam	20727	54	m	dyspepsia	gastritis	gastritis	AG	AG	P	P
40	prabhu	20732	26	m	abdominal pain	gastritis	normal	SG	NORMAL	P	N
41	sofia	20513	23	f	dyspepsia	gastritis	gastritis	AG	SG	P	P
42	jeyaraman	20522	70	m	dyspepsia	gastritis	normal	AG	NOMAL	P	N
43	yamuna	20115	38	f	epigastric pain	gastritis	normal	AG	NOMAL	P	N
44	nagavalli	20811	34	f	dyspepsia	gastritis	gastritis	AG	AG	P	P
45	balu	19700	57	m	epigastric pain	normal	normal	NORMAL	NORMAL	N	N
46	thiagarajan	21621	40	m	dyspepsia	gastritis	gastritis	AG	SG	P	P
47	deenathayalan	20939	46	m	abdominal pain	gastritis	normal	SG	NORMAL	P	N
48	palaniammal	21699	60	f	dyspepsia	gastritis	normal	SG	NORMAL	P	N
49	mani	20904	45	m	abdominal pain	gastritis	gastritis	AG	AG	P	P
50	raja	21215	45	m	epigastric pain	gastritis	normal	SG	NORMAL	P	N
51	vijayalakshmi	21754	44	f	epigastric pain	gastritis	gastritis	AG	SG	P	P
52	indrani	21751	55	f	dyspepsia	gastritis	normal	AG	NOMAL	P	N
53	rahmath	21771	33	f	epigastric pain	gastritis	normal	AG	NOMAL	P	N
54	sivananthan	21892	40	m	dyspepsia	gastritis	gastritis	AG	AG	P	P
55	govindsamy	18666	60	m	abdominal pain	normal	normal	NORMAL	NORMAL	N	N
56	eegan	20740	56	m	dyspepsia	gastritis	gastritis	AG	SG	P	P
57	sheela	21807	50	f	dyspepsia	gastritis	normal	SG	NORMAL	P	N
58	gangan	27073	46	m	epigastric pain	gastritis	normal	SG	NORMAL	P	N
59	jeyasree	22084	42	m	dyspepsia	gastritis	gastritis	AG	AG	P	P
60	sreenivasan	22206	42	m	epigastric pain	gastritis	normal	SG	NORMAL	P	N